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0014810140 BIOSIS NO.: 200400190897

Invasion of porcine brain microvascular endothelial cells by

Streptococcus ***suis*** serotype 2.

AUTHOR: Vanier Ghyslaine; Segura Mariela; Friedl Peter; Lacouture Sonia;
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JOURNAL: Infection and Immunity 72 (3): p1441-1449 March 2004 2004

MEDIUM: print

ISSN: 0019-9567 (ISSN print)

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: **Streptococcus suis** is an important swine pathogen

that mainly causes meningitis and occasionally causes other infections,
such as endocarditis, arthritis, and pneumonia. The pathogenesis of

S . ***suis*** infection has not been completely defined. However,
in order to cause meningitis, ***S*** . ***suis*** has to cross the
blood-brain barrier (BBB) made up of brain microvascular endothelial
cells. The objective of this work was to study the interactions of

S . ***suis*** serotype 2 with porcine brain microvascular
endothelial cells (PBMEC). The ability of North American and European

S . ***suis*** serotype 2 strains to adhere to PBMEC and, most
importantly, to invade PBMEC was demonstrated by using an antibiotic
protection assay and was confirmed by electron microscopy. The
polysaccharide ***capsule*** of ***S*** . ***suis*** seemed to

partially

interfere with the adhesion and invasion abilities of the bacterium. Our
results showed that intracellular viable ***S*** . ***suis*** could be
found in PBMEC up to 7 h after antibiotic treatment. Inhibition studies
demonstrated that invasion of PBMEC by ***S*** . ***suis*** required actin
microfilaments but not microtubular cytoskeletal elements or active
bacterial RNA or protein synthesis. At high bacterial doses,
suilysin-positive strains were toxic for PBMEC. The role of suilysin in
cytotoxicity was confirmed by using purified suilysin, electron
microscopy, and the lack of toxicity of a suilysin-negative mutant. In
swine, the invasion of endothelial cells of the BBB could play an
important role in the pathogenesis of the meningitis caused by ***S*** .

suis .

0011639781 BIOSIS NO.: 199800434028

Streptococcus suis and group B *Streptococcus* differ in their interactions with murine macrophages

AUTHOR: Segura Mariela A; Cleroux Patrick; Gottschalk Marcelo (Reprint)

AUTHOR ADDRESS: Groupe Rech. Maladies Infect. Porc, Fac. Med. Vet., Univ.

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JOURNAL: FEMS Immunology and Medical Microbiology 21 (3): p189-195 July, 1998 1998

MEDIUM: print

ISSN: 0928-8244

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: **Streptococcus suis** type 2 and group B *Streptococcus* type III (GBS) are important encapsulated bacterial species causing meningitis. In the present study we compared quantitatively the uptake and intracellular survival of ***S*** . ***suis*** type 2 and GBS type III with murine macrophages in non-opsonic conditions. The role of the **capsule** of both pathogens was also studied using previously obtained unencapsulated isogenic mutants. Encapsulated ***S*** . ***suis*** wild-type strain was practically not phagocytosed, while the unencapsulated mutant was easily ingested by macrophages. On the other hand, the well encapsulated GBS strain and its unencapsulated mutant were both phagocytosed in large numbers. Even if ***S*** . ***suis*** unencapsulated mutant showed a higher uptake rate than the parental strain, this value was always markedly lower than the numbers of ingested GBS strains. In addition, the intracellular survival of encapsulated and unencapsulated GBS strains was significantly higher than that of ***S*** . ***suis*** strains. These results suggest that interactions between GBS type III and ***S*** . ***suis*** type 2 with murine macrophages as well as the role of the **capsule** as an antiphagocytic factor are different for the two bacterial pathogens.

0011370845 BIOSIS NO.: 199800165092

Streptococcus suis serotype 2 mutants deficient in
capsular expression

AUTHOR: Charland Nathalie; Harel Josee; Kobisch Marylene; Lacasse Serge;
Gottschalk Marcelo (Reprint)

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JOURNAL: Microbiology (Reading) 144 (2): p325-332 Feb., 1998 ***1998***

MEDIUM: print

ISSN: 1350-0872

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: **Streptococcus suis** serotype 2 is responsible for a wide variety of porcine infections. In addition, it is considered a zoonotic agent. Knowledge about the virulence factors for this bacterium is limited but its polysaccharide **capsule** is thought to be one of the most important. Transposon mutagenesis with the self-conjugative transposon Tn916 was used to obtain acapsular mutants from the virulent ***S*** . ***suis*** type 2 reference strain S735. Clones were screened by colony-dot ELISA with a monoclonal antibody specific for a type 2 **capsular** epitope and clones that failed to react with the antibody were characterized. Two mutants, 2A and 79, having one and two Tn916 insertions respectively, were chosen for further characterization. Absence of **capsule** was confirmed by coagglutination, capillary precipitation and **capsular** reaction tests and by transmission electron microscopy. Absence of ***capsular*** polysaccharides correlated with increased hydrophobicity and phagocytosis by both murine macrophages and porcine monocytes compared to the wild-type strain. Furthermore, both mutants were shown to be avirulent in murine and pig models of infection. Finally, mutant 2A was readily eliminated from circulation in mice compared to the wild-type strain, which persisted more than 48 h in blood. Thus, isogenic mutants defective in ***capsule*** production demonstrate the importance of **capsular** polysaccharides as a virulence factor for ***S*** . ***suis*** type

0010946926 BIOSIS NO.: 199799580986

Production and characterization of **Streptococcus suis** type 2
mutants deficient in **capsular** expression

AUTHOR: Charland N (Reprint); Harel J (Reprint); Kobisch M; Jacques M
(Reprint); Gottschalk M (Reprint)

AUTHOR ADDRESS: Faculty Veterinary Med., Univ. Montreal, St-Hyacinthe, PQ,
Canada**Canada

JOURNAL: Abstracts of the General Meeting of the American Society for
Microbiology 97 (0): p37 1997 **1997**

CONFERENCE/MEETING: 97th General Meeting of the American Society for
Microbiology Miami Beach, Florida, USA May 4-8, 1997; 19970504

ISSN: 1060-2011

DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster

RECORD TYPE: Citation

LANGUAGE: English

0010519529 BIOSIS NO.: 199699153589

Role of **capsular** sialic acid in virulence and resistance to phagocytosis of **Streptococcus suis capsular** type 2

AUTHOR: Charland Nathalie; Kobisch Marylene; Martineau-Doize Beatrice; Jacques Mario; Gottschalk Marcelo (Reprint)

AUTHOR ADDRESS: Groupe Rech. Maladies Infect. Porc, Fac. Med. Vet., Univ. Montreal, CP 5000, St. Hyacinthe, PQ J2S 7C6, Canada**Canada

JOURNAL: FEMS Immunology and Medical Microbiology 14 (4): p195-203 1996 1996

ISSN: 0928-8244

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: **Streptococcus suis capsular** type 2 has a

capsule rich in sialic acid (NANA). Sialic acid, known to be an antiphagocytic factor for many bacterial species, inhibits the activation of the alternative complement pathway. The role of ***capsular*** NANA in virulence, resistance to phagocytosis and intracellular survival of

S . ***suis*** ***capsular*** type 2 was evaluated. In general, a low concentration of NANA was observed for all the ***S*** . ***suis*** strains tested. In addition, no difference could be found in NANA concentrations between strains of different virulence degrees. Sialic acid concentration increased in the virulent strain 89-1591 and the avirulent strain 90-1330 after in vivo growth with in increased

capsular material thickness compared to growth in vitro. No significant difference could be found in the phagocytosis rate by porcine blood monocytes of either strain and strain 89-1591 treated with sialidase or the sialic acid-binding, lectin from Sambucus nigra (SNA I). Intracellular survival of strain 89-1591 decreased after treatments with sialidase or lectin. becoming comparable to that of strain 90-1330.

Finally, no difference could be seen in virulence using a murine model. even if strain 89-1591 was treated with the enzyme or the lectin. Thus, NANA does not seem to be a critical virulence factor for ***S*** .

suis ***capsular*** type 2.

8212317 Genuine Article#: 258NC Number of References: 21

Title: Hybridization analysis of the gene encoding a hemolysin (suilysin) of *Streptococcus suis* type 2: evidence for the absence of the gene in some isolates

Author(s): Okwumabua O (REPRINT) ; Abdelmagid O; Chengappa MM

Corporate Source: TUSKEGEE UNIV, COLL VET MED NURSING & ALLIED HLTH, DEPT PATHOBIOL/TUSKEGEE//AL/36088 (REPRINT); KANSAS STATE UNIV, COLL VET MED, DEPT PATHOBIOL DIAGNOST MED/MANHATTAN//KS/66506

Journal: FEMS MICROBIOLOGY LETTERS, 1999, V181, N1 (DEC 1), P113-121

ISSN: 0378-1097 Publication date: 19991201

Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS

Language: English Document Type: ARTICLE

Abstract: A hemolysin gene was cloned from a virulent strain of

Streptococcus ***suis*** type 2 strain 1933. Analysis of the gene and its product revealed that it is identical to a previously reported hemolysin (suilysin) of ***S*** . ***suis*** type 2. Southern hybridization analysis of the digested total genomic DNA from ***S*** . *suis* with the cloned hemolysin DNA sequences as probe indicated that the hemolysin gene is present as a single copy on the genome. Genomic DNA of 63 isolates of ***S*** . ***suis*** encompassing all known serotypes were examined by DNA hybridization and polymerase chain reaction (PCR) studies for the presence of the hemolysin gene homolog. The results of both techniques were identical and demonstrated the absence of the hemolysin gene in some isolates. In DNA hybridization studies, three DNA probes derived from the hemolysin encoding gene were used. Results showed that sequences encoding the C-terminal 257 amino acid residues (Probe 1) were the most conserved and hybridized to a 1.2 kb fragment in 32 (51%) strains and a 4.0 kb fragment in 23 (36%) strains respectively. Thus, Probe 2 hybridized to the DNA of 55 (87%) of the isolates tested. The first probe (Probe 1) comprising almost the entire hemolysin gene and the third probe (Probe 3) which consisted of the N-terminal sequences hybridized only to a 4.0 kb fragment in 23 (36%) of the strains tested. Eight (13%) of the strains tested were hybridization and PCR negative. The hybridization of the C-terminal end sequences (Probe 2) to the 1.2 kb fragment in 32 (51%) of the strains and the lack of hybridization of the probes to eight (13%) strains may suggest the presence of different types of hemolysin molecule in

S . ***suis*** strains. (C) 1999 Published by Elsevier Science B.V. All rights reserved.

0015055074 BIOSIS NO.: 200400425863

Encapsulated **Streptococcus suis** inhibits activation of signaling pathways involved in phagocytosis

AUTHOR: Segura Mariela; Gottschalk Marcelo; Olivier Martin (Reprint)

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JOURNAL: Infection and Immunity 72 (9): p5322-5330 September 2004 2004

MEDIUM: print

ISSN: 0019-9567 (ISSN print)

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: **Streptococcus suis capsular** type 2 is an

important zoonotic agent of meningitis. Previous studies reported that, in contrast to nonencapsulated mutants, encapsulated ***S*** . ***suis*** is able to resist phagocytosis. However, the mechanisms by which ***S*** . ***suis*** avoids phagocytosis are unknown. To elucidate the signaling pathway(s) involved in ***S*** . ***suis*** antiphagocytosis, we compared the ability of an encapsulated strain and its nonencapsulated mutant to induce the activation of Akt and protein kinase C (PKC), which are downstream kinases of the phosphatidylinositol 3-kinase (PI-3K) pathway, known to be involved in the phagocytosis processes. The results demonstrated high levels of Akt and PKC α phosphorylation after infection of J774 macrophages with the nonencapsulated mutant, whereas the encapsulated strain showed reduced activation of PI-3K/Akt/PKC α signaling pathway, as well as several protein tyrosine events. These results correlated with the number of intracellular bacteria. Macrophages pretreated with specific PI-3K or PKC inhibitors showed reduced levels of Akt and PKC α phosphorylation, resulting in 50% reduction of phagocytosis. The role of phosphatases in the antiphagocytic mechanisms was evaluated by using phosphatase inhibitors, as well as SHP-1-deficient macrophages. Only in the absence of SHP-1 did the phagocytosis of encapsulated ***S*** . ***suis*** significantly increase, leading to Akt phosphorylation levels similar to those observed with the nonencapsulated strain, indicating activation of this important SH2 domain-containing tyrosine phosphatase by encapsulated ***S*** . ***suis*** . Finally, when purified ***S*** . ***suis*** ***capsular*** polysaccharide (CPS) was added to macrophages, no phosphorylation events were observed. In addition, CPS and encapsulated ***S*** . ***suis*** were able to inhibit the uptake of the nonencapsulated mutant. These results suggest the importance of CPS in the mechanisms, whereby ***S*** . ***suis*** downmodulates phagocytosis.

knock-out (nok'out)

A genetically engineered organism in which the genome has been altered by site-directed recombination so that a gene is deleted.

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<input type="checkbox"/>	L2	(s adj1 suis) or (streptococcus adj1 suis)	585
<input type="checkbox"/>	L1	streptococcus same suis	465

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